GLP-1 Drugs, Insulin and Visceral Fat: The Paradox That Isn’t

At first glance, GLP-1 receptor agonists appear to contradict one of the most basic principles of metabolism.

They stimulate insulin secretion and improve insulin sensitivity — yet they are among the most effective therapies for reducing **visceral adipose tissue (VAT)**, a fat depot that accumulates largely as a consequence of **chronic insulin exposure**.(1)

How can drugs that enhance insulin action lead to loss of insulin-driven fat?

The answer is that GLP-1 therapies do not exaggerate pathological insulin signalling. Instead, they **restore physiological insulin behaviour**, reduce inappropriate insulin exposure over time, and dismantle the metabolic environment in which visceral fat thrives.

Once this distinction is made, the apparent paradox disappears.

A Brief History: Why GLP-1 Drugs Were Developed

GLP-1 (glucagon-like peptide-1) is an incretin hormone released from the gut in response to food intake. Its physiological actions include stimulation of insulin secretion, suppression of glucagon, delayed gastric emptying, and promotion of satiety(2).

In the 1990s, it became clear that people with type 2 diabetes had an **impaired incretin effect**, contributing to post-prandial hyperglycaemia(3). This led to the development of **GLP-1 receptor agonists** as glucose-lowering therapies.

Early agents such as **exenatide** and **liraglutide** were tested primarily in people with diabetes, with glycaemic control as the main endpoint(4). Weight loss was not the primary objective and was initially regarded as a secondary effect.

That view changed as the magnitude, durability and metabolic quality of the weight loss became clear.

The “Viagra Moment”: Directionally True, Scientifically Instructive

GLP-1 drugs are often said to have had a “Viagra moment” — a point at which their unexpected effects revealed a much broader therapeutic potential.

While not a strict repurposing story, the analogy is **directionally correct**.

GLP-1 receptor agonists were always licensed for diabetes. However, the **extent of weight loss**, and in particular the **preferential reduction in visceral and hepatic fat**, could not be explained by glucose lowering alone(5).

This led to:

- higher-dose studies
- trials in non-diabetic populations
- and ultimately separate obesity licences (e.g. liraglutide 3.0 mg, semaglutide 2.4 mg)(6–8)

In retrospect, these drugs were not reinvented — their **true cardiometabolic reach was initially underestimated**.

The Core Misconception: “GLP-1 Drugs Increase Insulin”

It is correct that GLP-1 receptor agonists stimulate insulin secretion.
It is also the source of confusion.

Glucose-dependent insulin secretion

GLP-1 drugs stimulate insulin secretion **only when glucose levels are elevated** (9). When glucose normalises, insulin secretion falls. When glucose is low, GLP-1 has little or no insulin-stimulating effect.

This is fundamentally different from the **chronic basal hyperinsulinaemia** seen in insulin resistance, where insulin remains elevated even during fasting and between meals(10) Importantly, the pathological “insulin problem” in metabolic disease is typically not excessive physiological post-meal insulin responses per se, but **prolonged and repeated insulin exposure driven by insulin resistance (especially skeletal muscle), impaired metabolic switching, and hepatic lipid overflow**.(11)

An Upstream Driver: Skeletal Muscle Loss as the Initiating Event

An important and often underappreciated upstream contributor to this entire metabolic cascade may be **age-related loss of skeletal muscle mass and function**.

Skeletal muscle is the principal site of insulin-mediated glucose disposal, accounting for approximately **70–80% of post-prandial glucose uptake** in healthy individuals. Preservation of muscle mass and muscle insulin sensitivity therefore provides a powerful metabolic "sink" for glucose following carbohydrate intake. When this capacity is reduced, post-prandial glucose excursions are prolonged and compensatory insulin secretion increases.(1,10)

In mid-life, several converging factors act to reduce skeletal muscle mass and quality:

- **Reduced physical activity and resistance loading**, particularly loss of upper-body and type II muscle fibres, leading to reduced insulin-mediated glucose uptake(10,12)
- **Age-related anabolic resistance**, whereby muscle becomes less responsive to dietary protein and exercise stimuli, accelerating net muscle loss and impairing glucose disposal(12)
- **Inadequate dietary protein intake or poor protein distribution**, increasing reliance on skeletal muscle catabolism to supply amino acids for tissue repair and immune function, with gradual loss of lean mass(12)
- In this context, amino acids required for ongoing cellular and tissue maintenance may increasingly be derived from **catabolism of existing skeletal muscle**, resulting in slow, often clinically silent muscle loss. This reduces total muscle glucose-disposal capacity even before overt sarcopenia becomes apparent.

The metabolic consequence is a **loss of buffering capacity for post-prandial glucose**. After carbohydrate intake, glucose remains elevated for longer, provoking prolonged and repeated insulin secretion to maintain glycaemic control. Over time, this leads to **chronic hyperinsulinaemia driven primarily by skeletal muscle insulin resistance**, rather than excessive caloric intake alone.[10],[22]

Muscle Loss as the Trigger for Lipid Spillover and VAT Accumulation

Once muscle glucose uptake is impaired, the downstream sequence accelerates:

- **Prolonged post-prandial hyperglycaemia**, sustaining insulin secretion[10]
- **Adipose tissue insulin resistance**, reducing insulin's ability to suppress lipolysis and increasing circulating free fatty acid flux[10]

- **Hepatic exposure to excess glucose and fatty acids**, driving triglyceride synthesis and increased VLDL export[2]
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- When subcutaneous adipose tissue reaches its limit of healthy expansion, surplus energy is redirected into ectopic depots, including hepatic and visceral fat, consistent with the **adipose tissue expandability hypothesis**.

In this framework, visceral adipose tissue accumulation is not the primary defect, but a **downstream consequence of reduced metabolic capacity**, beginning with loss of skeletal muscle mass and function. VAT then feeds back negatively via portal fatty acid delivery and inflammatory signalling, further impairing hepatic and systemic insulin sensitivity and accelerating cardiometabolic risk.

This integrated model helps explain why metabolic disease often emerges in mid-life, why waist circumference may increase despite relatively stable body weight, and why early disease is frequently missed by standard biochemical screening.

Implications for GLP-1 Therapy and Long-Term Strategy

GLP-1 receptor agonists do not directly reverse skeletal muscle loss. However, by reducing glucose load, smoothing post-prandial glucose delivery, lowering chronic insulin exposure, suppressing hepatic glucose output, and reducing ectopic lipid spillover, they **remove the metabolic pressure created by reduced muscle glucose-disposal capacity**.

This allows visceral and hepatic fat to mobilise even in the presence of reduced muscle mass, explaining the early and preferential reduction in VAT observed with GLP-1 therapy.(13) Over time, as VAT and liver fat decline, inflammatory tone falls and insulin sensitivity improves, partially restoring metabolic flexibility.

Critically, this framework highlights why **preservation and rebuilding of skeletal muscle**—through resistance training and adequate dietary protein—should be regarded as **foundational metabolic therapy**, particularly in mid-life and beyond. GLP-1 pharmacotherapy and muscle-directed lifestyle intervention are therefore **complementary strategies acting on different levels of the same biological system**.

Muscle Loss as the Trigger for Lipid Spillover and VAT Accumulation

Once muscle glucose uptake is impaired, the downstream sequence described earlier accelerates:

- prolonged post-prandial hyperglycaemia sustains insulin secretion
- adipose tissue becomes progressively resistant to insulin's anti-lipolytic effect
- circulating free fatty acid flux increases
- the liver is exposed to excess glucose and fatty acids, increasing triglyceride synthesis and VLDL export

When subcutaneous adipose tissue reaches its limit of healthy expansion, surplus energy is redirected into ectopic depots, including hepatic and visceral fat, consistent with the adipose tissue expandability hypothesis(1).

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Importantly, this framework underscores why **preservation and rebuilding of skeletal muscle**—through resistance training and adequate dietary protein—should be viewed not as an optional adjunct, but as **foundational metabolic therapy**, particularly in mid-life and beyond. GLP-1 pharmacotherapy and muscle-directed lifestyle intervention are therefore best understood as **complementary rather than competing strategies**.

Chronic Insulin Exposure and Lipid Spillover Drives Visceral Fat Accumulation

Visceral adipose tissue does not accumulate because of brief physiological insulin spikes. It expands gradually over **days to weeks** when the metabolic system is repeatedly pushed into a prolonged post-prandial state and insulin is seldom allowed to fall. In most individuals this environment is initiated and sustained by **skeletal muscle insulin resistance**, which impairs glucose disposal after carbohydrate intake. Post-prandial glucose therefore remains elevated for longer, provoking compensatory and repeated insulin secretion to maintain glycaemic control.(10)

In parallel, adipose tissue becomes less responsive to insulin's anti-lipolytic action, increasing circulating free fatty acid flux. The liver, exposed to excess glucose and fatty acids, increases triglyceride synthesis and exports lipid as VLDL-triglyceride, thereby amplifying systemic lipid delivery. When the "safe" subcutaneous adipose depot reaches its limit of healthy expansion (reduced adipose expandability), surplus energy is diverted into ectopic depots, including hepatic and visceral fat. This overflow biology helps explain why VAT accumulation often represents **adipose dysfunction and ectopic fat partitioning**, rather than simple weight gain.(1) VAT is frequently **insulin resistant** in this state and relatively more lipolytic than subcutaneous fat, yet remains metabolically harmful because of its portal drainage and inflammatory signalling. Over time, VAT therefore becomes both a marker of systemic insulin resistance and ectopic lipid overflow, and a contributor to worsening hepatic insulin resistance and cardiometabolic risk.(10,11)

Why GLP-1 Therapy Reduces Total Insulin Exposure

1. Appetite Suppression and Reduced Post-Prandial Insulin Demand

GLP-1 receptors in the hypothalamus and brainstem reduce hunger, food reward and hedonic eating(14).

As a result, patients typically:

- eat fewer calories
- snack less
- reduce portion sizes without conscious restraint

This reduction in caloric intake leads to **fewer and smaller post-prandial glucose excursions**, which in turn reduces **post-prandial insulin requirements**. With GLP-1 therapy, insulin secretion remains glucose-dependent — rising appropriately when glucose rises, but not overshooting when glucose exposure is reduced.

The net effect is **lower post-prandial insulin exposure**, while maintaining or improving post-prandial glucose control.

2. Slowing of gastric emptying and modulation of portal glucose delivery

By delaying gastric emptying, carbohydrate-containing food enters the small intestine more gradually, resulting in **slower absorption of glucose** into the circulation. This reduces the rate at which glucose is delivered to the liver via the portal vein and blunts early post-prandial glucose excursions. Because insulin secretion is primarily driven by rising plasma glucose, this slower glucose delivery is associated with **smaller and more physiologically timed insulin responses**, particularly in the early post-prandial period(15,16). The magnitude of the gastric emptying effect differs between agents. It is most pronounced with short-acting GLP-1 receptor agonists and attenuated with long-acting agents such as semaglutide due to tachyphylaxis. Nevertheless, even with long-acting preparations, clinically meaningful reductions in early post-prandial glucose excursions are consistently observed.(16)

Visceral adipose tissue, via its portal drainage and proximity to hepatic metabolism, sits within a physiology where repeated rapid portal glucose delivery and compensatory insulin secretion contribute to chronic suppression of lipolysis and progressive ectopic lipid partitioning over days to weeks. In addition to their effects on appetite and glucose-dependent insulin secretion, GLP-1 receptor agonists exert an important influence on post-prandial glucose handling through **slowing of gastric emptying**. This is a physiological action of native GLP-1 and is retained, to varying degrees, by GLP-1 receptor agonists.(3,15)

This effect complements appetite reduction, reduced carbohydrate intake, suppression of hepatic glucose output, and improved insulin sensitivity, reinforcing the central concept that GLP-1 receptor agonists reduce visceral fat not by increasing insulin exposure, but by **normalising insulin timing and reducing inappropriate insulin signalling over time**.

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3. Suppression of Hepatic Glucose Output

Basal insulin levels are strongly influenced by hepatic glucose production.

GLP-1 receptor agonists:

- suppress inappropriate glucagon secretion
- improve hepatic insulin sensitivity
- reduce endogenous glucose production(17)

As fasting glucose levels fall, the requirement for compensatory basal insulin secretion diminishes — an important mechanism in reducing visceral fat accumulation.

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4. Preferential Reduction in Visceral and Hepatic Fat

Imaging studies consistently show that GLP-1 receptor agonists reduce visceral fat and liver fat disproportionately compared with subcutaneous fat.(13,16) From a mechanistic standpoint, this is best understood not as “VAT being insulin sensitive”, but as **VAT being a dynamic ectopic depot** that expands and regresses in response to the chronic metabolic environment.

In insulin-resistant states, repeated carbohydrate-driven hyperglycaemia and compensatory hyperinsulinaemia, together with increased free fatty acid flux and hepatic VLDL-triglyceride export, promote progressive deposition into liver and visceral depots—particularly once subcutaneous adipose storage capacity is constrained. When GLP-1 therapy reduces caloric intake, smooths post-prandial glucose delivery, suppresses hepatic glucose output, and improves insulin sensitivity, the chronic drivers of ectopic lipid partitioning diminish. As a result, hepatic fat and VAT are often mobilised early, with waist circumference frequently improving before dramatic changes in total body weight are seen.

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5. Improved Insulin Sensitivity Through Fat Loss

Visceral adipose tissue releases inflammatory cytokines and free fatty acids that impair insulin signalling(13).

As VAT reduces:

- inflammatory tone falls
- skeletal muscle insulin sensitivity improves
- hepatic insulin sensitivity improves

Over time, this leads to **reductions in both basal and post-prandial insulin requirements**, particularly in people with type 2 diabetes, and also observed in type 1 diabetes following significant weight loss.

Cardiovascular Outcomes: Semaglutide in Context

The metabolic improvements associated with GLP-1 therapy translate into **hard cardiovascular outcomes**.

In 2023, the SELECT trial demonstrated that **semaglutide 2.4 mg weekly reduced major adverse cardiovascular events (MACE) by ~20%**, with an **absolute risk reduction of ~1.5% over 3.3 years**, giving a **number needed to treat (NNT) of approximately 67** in people with established cardiovascular disease **without diabetes**(18).

This places semaglutide firmly in the realm of **disease-modifying cardiovascular therapy**.

To put this into clinical perspective, it is helpful to compare the magnitude of benefit with that of statins — the cornerstone of secondary prevention.

Comparative Cardiovascular Benefit: Semaglutide vs Statins

Feature	Semaglutide (SELECT)	Statins (secondary prevention)
Population	Overweight/obese patients with established CVD, **no diabetes**	Patients with established CVD
Follow-up	~3.3 years	Typically ~5 years
Primary endpoint	MACE (CV death, MI, stroke)	MACE / coronary death
Relative risk reduction	~20%	~20–30%
Absolute risk reduction	~1.5%	~2–3%
NNT	~67 (3.3 years)	~33–50 (5 years)
Effect on CV death	Reduced	Reduced
Effect on MI	Reduced	Reduced
Effect on stroke	Reduced	Reduced
Primary mechanism	↓ VAT, ↓ inflammation, improved insulin biology	↓ LDL-C, plaque stabilisation
Effect on weight/VAT	Marked VAT reduction	Neutral or slight gain

Key point:
These therapies act through **different biological pathways** and their benefits are **additive, not competitive**.

Not a Paradox — a Physiological Reset

Seen superficially, it appears contradictory that a drug enhancing insulin action reduces insulin-driven fat.

Seen physiologically, it is entirely coherent.

GLP-1 receptor agonists:

- restore glucose-dependent insulin secretion
- reduce excessive post-prandial insulin exposure
- lower basal insulin requirements over time

- suppress appetite
- reduce hepatic glucose output
- dismantle the VAT–insulin resistance cycle

The net effect is a metabolic environment that favours **fat mobilisation rather than fat storage**.

A final thought: insulin, visceral fat and cancer risk

There is growing evidence that the same biology linking visceral fat to heart disease may also be relevant to **cancer risk**.

Visceral fat is not just an energy store — it is hormonally and metabolically active. It is associated with **chronically raised insulin levels**, increased insulin-like growth factor (IGF-1) signalling, low-grade inflammation, and impaired immune surveillance. Together, these factors can create an internal environment that favours **cell proliferation and reduced apoptosis**, mechanisms that are biologically plausible contributors to cancer development.

Large population studies support this link. A landmark **Lancet meta-analysis** of prospective studies showed that excess body fat is associated with an increased risk of several common cancers, including cancers of the colon, breast (post-menopausal), pancreas, kidney and oesophagus. Importantly, this analysis highlighted that the relationship goes beyond simple body weight and is likely driven by underlying metabolic and hormonal factors, including insulin and growth-factor signalling(11).

More recently, an important study from Iceland (the AGES–Reykjavik cohort) used CT scanning to directly measure **visceral adipose tissue**, rather than relying on BMI. This study found that **higher levels of visceral fat were independently associated with increased cancer risk**, even after adjusting for overall body size. In other words, *where fat is stored* appeared to matter more than *how much someone weighed*(19).

Together, these findings help explain why some people with only modest weight gain — but high levels of visceral fat — may still carry increased cancer risk, while others with a higher BMI but predominantly subcutaneous (pinchable) fat may not.

While GLP-1 therapies are **not cancer treatments**, their ability to reduce visceral fat and chronic insulin exposure raises important questions about whether improving this metabolic environment could, over the long term, reduce risk not only of heart disease and diabetes, but also of certain obesity-related cancers. This remains an active area of research, but it reinforces a broader message: targeting visceral fat is about far more than weight — it is about **reducing systemic biological stress on the body**.

Summary

GLP-1 drugs do not cause fat loss *despite* insulin.

They cause fat loss because they **end chronic, inappropriate insulin exposure while preserving insulin's essential, time-limited role**.

What looks like a paradox is, in reality, a restoration of metabolic physiology closer to its evolutionary design.

And visceral fat — dependent on constant insulin signalling — shrink away in that environment.

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